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Impact of Coronary Plaque Composition on Cardiac Troponin Elevation After Percutaneous Coronary Intervention in Stable Angina Pectoris

A Computed Tomography Analysis

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Objectives The authors used multidetector computed tomography (MDCT) to study the relation between culprit plaque characteristics and cardiac troponin T (cTnT) elevation after percutaneous coronary intervention (PCI).

Background Percutaneous coronary intervention is often complicated by post-procedural myocardial necrosis manifested by elevated cardiac biomarkers.

Methods Stable angina patients ($n = 107$) with normal pre-PCI cTnT levels underwent 64-slice MDCT before PCI to evaluate plaque characteristics of culprit lesions. Patients were divided into 2 groups according to presence (group I, $n = 36$) or absence (group II, $n = 71$) of post-PCI cTnT elevation $\geq$3 times the upper limit of normal (0.010 ng/ml) at 24 h after PCI.

Results Computed tomography attenuation values were significantly lower in group I than in group II (43.0 [26.5 to 75.7] HU vs. 94.0 [65.0 to 109.0] HU, $p < 0.001$). Remodeling index was significantly greater in group I than in group II (1.20 ± 0.18 vs. 1.04 ± 0.15, $p < 0.001$). Spotty calcification was observed significantly more frequently in group I than in group II (50% vs. 11%, $p < 0.001$). Multivariate analysis showed presence of positive remodeling (remodeling index >1.05; odds ratio: 4.54; 95% confidence interval: 1.36 to 15.9; $p = 0.014$) and spotty calcification (odds ratio: 4.27; 95% confidence interval: 1.30 to 14.8; $p = 0.016$) were statistically significant independent predictors for cTnT elevation. For prediction of cTnT elevation, the presence of all 3 variables (CT attenuation value <55 HU; remodeling index >1.05, and spotty calcification) showed a high positive predictive value of 94%, and their absence showed a high negative predictive value of 90%.

Conclusions MDCT may be useful in detecting which lesions are at high risk for myocardial necrosis after PCI. (J Am Coll Cardiol 2012;59:1881–8) © 2012 by the American College of Cardiology Foundation

Percutaneous coronary intervention (PCI) may be complicated by post-procedural myocardial injury/infarction as manifested by elevated cardiac biomarkers such as creatine kinase-myocardial band or troponin T. The occurrence of post-procedural myocardial injury/infarction has been shown to be associated with worse short- and long-term clinical outcome (1,2), and even mild elevation of cardiac troponin after PCI has been related to a worse prognosis (3). Intravascular ultrasonography (IVUS) has shown that post-procedural myocardial injury/infarction is caused by lesions with ruptured plaques and/or those with a greater plaque burden (4). Virtual histology IVUS also has shown that post-PCI cardiac troponin (cTnT) elevation occurs in lesions with a large necrotic core area and positive remodeling (PR) of the vessel (5).

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Multidetector computed tomography (MDCT) is a promising technique for noninvasive coronary angiography. With the development of MDCT, it is possible not only to detect coronary artery stenosis (6,7) but also to evaluate coronary plaque quality and quantity such as can be done with IVUS (8,9). Motoyama et al. (10) showed that the

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Abbreviations and Acronyms

ACS = acute coronary syndrome(s)
CSA = cross-sectional area
CT = computed tomography
cTnT = cardiac troponin T
EEM = external elastic membrane
IVUS = intravascular ultrasonography
MDCT = multidetector computed tomography
NPV = negative predictive value
PCI = percutaneous coronary intervention
PPV = positive predictive value
PR = positive vessel remodeling
RI = remodeling index
SAP = stable angina pectoris

computed tomography (CT) characteristics of culprit lesions in acute coronary syndrome (ACS) included PR, low-attenuation plaques, and spotty calcification, and that the patients with these 2 plaque characteristics of PR and low-attenuation plaques on CT angiography were at a higher risk of developing ACS than were patients without these characteristics during follow-up (11). Recently, Uetani et al. (12) reported that post-procedural myocardial injury/infarction was associated with the volume and fraction of low-attenuation plaques detected by MDCT. However, a previous study exploring potential prognostic predictors of cardiovascular events on MDCT showed that mixed lesions were associated with adverse events on follow-up (13). Thin-cap fibroatheromas on virtual histology IVUS were most prevalent in mixed plaques, suggesting a higher degree of vulnerability of these mixed plaques on MDCT (14).

Therefore, we hypothesized that the plaque with MDCT characteristics of low attenuation plaques, PR, and spotty calcification has the potential to predict an elevation of cTnT after PCI. The aim of this study was to investigate the clinical value of PR and spotty calcification in conjunction with low-attenuation plaques on MDCT to predict post-PCI cTnT elevation.

Methods

Study population. The study population comprised 107 patients who were diagnosed as having stable coronary artery disease by 64-slice MDCT before PCI from April 2009 to August 2010 at our institution. We included patients with stable angina pectoris (SAP) with normal pre-PCI cTnT levels (15) and excluded patients with ST-segment elevation myocardial infarction and unstable AP. We also excluded patients with severely calcified lesions, motion artifacts, previously stented lesions, and chronic total occlusions, for which coronary plaque quantity and degree of stenosis are difficult to evaluate by MDCT.

Study protocol. All patients underwent 64-slice MDCT to evaluate coronary plaque characteristics of the culprit lesion before PCI and serial measurements of cTnT before PCI and at 24 h post-procedure. According to cTnT blood test results, the patients were divided into 2 groups according to the presence (group I, n = 36) or absence (group II, n = 71) of post-PCI TnT elevation ≥3 times the upper limit of normal (0.010 ng/ml) at 24 h after PCI.

MDCT protocol. Scanning was performed with a Philips Brilliance-64 scanner (Philips Medical Systems, Cleveland, Ohio) with 64 × 0.625-mm detector configuration. Scanning was performed at 120 kV and 600 to 1,050 mA, 0.2 pitch, and with standard or sharp filters. Estimated effective radiation dose was 11 mSv. Reconstruction was routinely performed using a window centered at 75% of the R-R interval to coincide with left ventricular diastasis. A volume of 60 ml of contrast agent (iopamidol 370 mg/ml; Schering AG, Berlin, Germany) was injected intravenously at a rate of 4 ml/s. As soon as the signal density level in the ascending aorta reached a pre-defined threshold of 100 Hounsfield units (HU), acquisition of CT data and an electrocardiogram trace were automatically started during a 7-s to 9-s breath-hold. The patients were given oral metoprolol (20 mg) 1 h before the scheduled scan if their heart rate was >70 beats/min, and all patients received sublingual nitroglycerin (0.3 mg) 5 min before the scan.

MDCT analysis. Analysis of the scans was performed using a Brilliance Workspace 3-D workstation (Philips Medical Systems). Images were initially reconstructed at mid-diastolic phase (75% of R-R interval) of the cardiac cycle. In some cases, additional reconstructions were made at different time points of the R-R interval. Each scan was analyzed independently by 2 experienced readers unaware of the patient's identity, clinical presentation, biomarker analysis, and PCI procedure. Image display settings for lumen and plaque quantification were determined according to previously published data (16).

We measured the vessel diameter and lesion length, the cross-sectional area (CSA) of the external elastic membrane (EEM) and target lesion, and the lumen CSA of the proximal and distal vessel references using axial images and multiplanar reconstruction images. The CT density values of the culprit plaque were measured from at least 3 points, and averaged this. Remodeling index (RI) was defined as the EEM CSA of the target lesion divided by the average of the EEM CSAs of the proximal and distal references. We assessed adherent calcium deposits in or adjacent to each plaque by determining their presence or absence and morphology as follows according to previously described methods: diffuse, length of calcium burden ≥3/2 of vessel diameter and width ≥2/3 of vessel diameter; medium, length ≥3/2 of vessel diameter and width <2/3 of vessel diameter or length <3/2 of vessel diameter and width ≥2/3 of vessel diameter; and spotty, length <3/2 of vessel diameter and width <2/3 of vessel diameter (17). We classified the plaque into 3 types, namely, noncalcified, calcified, and mixed plaque (18). A ringlike enhancement was defined as either the presence of a ring of high attenuation around certain coronary artery plaques or the CT attenuation of a ring presenting higher than those of the adjacent plaque and no greater than 130 HU (19).

PCI procedures. All patients received treatment with aspirin (200 mg/day) and clopidogrel (75 mg/day) at least 24 h before the procedure. A glycoprotein IIb/IIIa receptor

Table 1 Clinical Characteristics of the Study Population

Characteristic	Group I (cTnT Elevation ≥3×) (n = 36)	Group II (cTnT Elevation <3×) (n = 71)	p Value
Age, yrs	64 ± 10	66 ± 9	0.219
Male	30 (83%)	54 (76%)	0.379
Hypertension	22 (61%)	54 (76%)	0.112
Dyslipidemia	30 (83%)	55 (77%)	0.472
Diabetes mellitus	17 (47%)	35 (49%)	0.671
Smoking	22 (61%)	50 (70%)	0.147
LDL cholesterol, mg/dl	100.4 ± 26.7	92.6 ± 22.9	0.151
HDL cholesterol, mg/dl	46.9 ± 17.0	49.5 ± 15.0	0.467
Triglyceride, mg/dl	137 (90.5–195)	129 (82.5–195)	0.802
C-reactive protein, mg/l	1.50 (0.65–3.50)	1.00 (0.60–2.50)	0.195
Medications			
Beta-blocker	12 (33%)	26 (37%)	0.737
ACEI/ARB	17 (48%)	34 (49%)	0.953
CCB	16 (45%)	44 (63%)	0.125
Statin	23 (64%)	53 (75%)	0.234

Values are mean ± SD, n (%), median (interquartile range).

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium-channel blocker; cTnT = cardiac troponin T; HDL = high-density lipoprotein; IQR = interquartile range; LDL = low-density lipoprotein.

inhibitor is not yet available in Japan. The PCI was performed through either the radial or femoral artery using a 6-F catheter. Before starting the procedure, 6,000 to 10,000 U of heparin were given intravenously, and the activated clotting time was maintained at >300 s. Once the guide wire passed through the culprit lesion, balloon dilation or stent placement was performed. The treatment was regarded as successful when the luminal diameter of the target lesion increased by at least by 50%, residual stenosis was less than 30%, and the TIMI (Thrombolysis In Myocardial Infarction) flow was grade 3 (20). Transient no reflow was defined as an angiogram showing a deterioration of coronary flow of TIMI grade 0, 1, or 2 during the procedure, regardless of the timing, and TIMI grade 3 at the final angiogram (21).

Statistical analysis. All data are expressed as the mean ± SD or median and interquartile range for non-normally distributed data. Comparisons of continuous variables were analyzed by unpaired *t* test or Mann-Whitney *U* test according to the data distribution. Comparisons of categor-

Table 2 Culprit Lesion and Procedural Characteristics

Characteristic	Group I (cTnT Elevation ≥3×) (n = 36)	Group II (cTnT Elevation <3×) (n = 71)	p Value
Target vessel			
LAD	21 (58%)	37 (52%)	0.541
RCA	7 (19%)	19 (26%)	0.398
LCX	8 (22%)	15 (21%)	0.896
Type B2/C lesion	16 (45%)	30 (43%)	0.789
Bifurcation	10 (28%)	19 (27%)	0.953
Procedural characteristics			
Number of stents	1.22 ± 0.50	1.14 ± 0.45	0.373
Stent size, mm	2.82 ± 0.36	2.79 ± 0.54	0.789
Transient no reflow	6 (16%)	2 (3%)	0.012
MDCT measurements			
Lesion EEM CSA, mm^2	15.6 ± 1.1	14.1 ± 0.8	0.252
Lesion MLA, mm^2	2.62 ± 1.44	2.88 ± 1.27	0.381
Lesion length, mm	16.7 ± 4.2	14.1 ± 5.1	0.012
Remodeling index	1.20 ± 0.18	1.04 ± 0.15	<0.001
Positive remodeling	29 (80%)	24 (32%)	<0.001
CT attenuation value, HU	43.0 (26.5–75.7)	94.0 (65.0–109.0)	<0.001
Ringlike enhancement	11 (31%)	8 (11%)	0.016

Values are n (%), mean ± SD, or median (interquartile range).

CSA = cross-sectional area; CT = computed tomography; cTnT = cardiac troponin T; EEM = external elastic membrane; HU = Hounsfield units; LAD = left anterior descending; LCX = left circumflex; MDCT = multidetector computed tomography; MLA = minimum lumen area; RCA = right coronary artery.

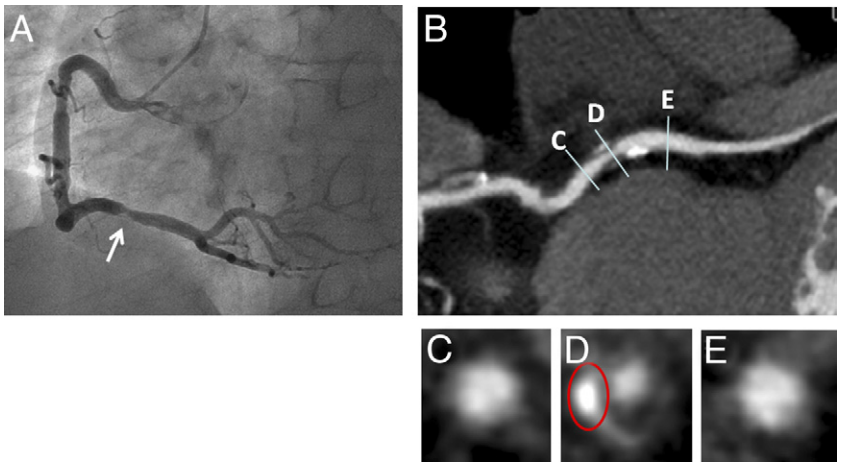

Figure 1 **CT Characteristics of Culprit Lesion in Patient With Post-PCI TnT Elevation ≥3 Times Upper Limit of Normal**

Computed tomography (CT) characteristics of culprit lesion **(arrow)** in a 52-year-old man with troponin T (TnT) elevation ≥3 times the upper limit of normal after percutaneous coronary intervention (PCI). **(A)** Coronary angiogram and **(B)** multiplanar reconstructed image show severe stenosis in the mid right coronary artery. Cross-sectional images show **(C)** the proximal reference, **(D)** the culprit lesion, and **(E)** the distal reference. The lesion has positive remodeling (remodeling index: 1.28), spotty calcification, and low CT density (16 HU). **Red circle** indicates area of spotty calcification.

ical variables between groups were performed by chi-square test without correction for multiplicity. Interobserver and intraobserver agreements were performed by linear regression analyses for continuous variables and kappa (κ) test for categorical variables. Receiver-operating characteristic analysis was used to determine optimal cutoff values of CT attenuation value and RI for prediction of post-PCI cTnT elevation. The best cutoff value was defined as the point with the highest sum of sensitivity and specificity. Results displayed for univariate analysis were based on clinically relevant and laboratory-based variables and lesion characteristics. Multivariate logistic regression analysis was then developed to calculate odds ratios and 95% confidence intervals. We performed stepwise forward selection considering any valuables with values of $p < 0.20$ to identify potential risk factors for post-PCI cTnT elevation. The sensitivity,

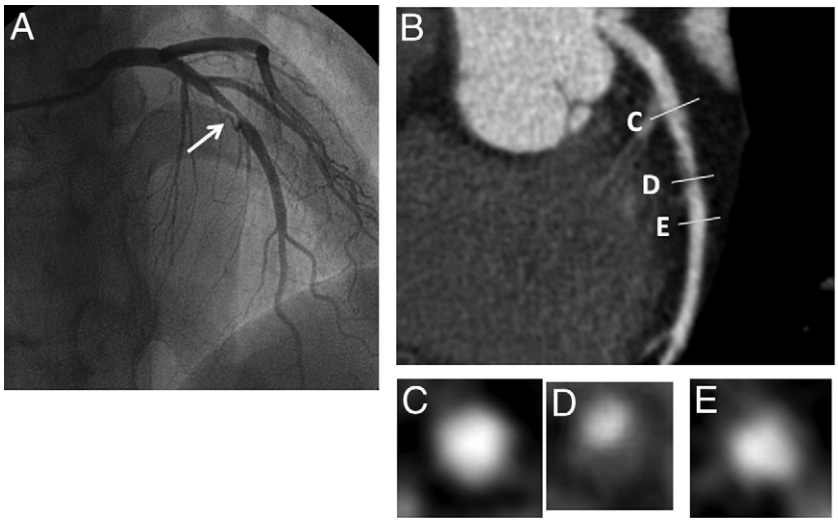

Figure 2 **CT Characteristics of Culprit Lesion in Patient Without Post-PCI TnT Elevation ≥3 Times Upper Limit of Normal**

Computed tomography (CT) characteristics of the culprit lesion **(arrow)** in a 51-year-old man without troponin T (TnT) elevation ≥3 times the upper limit of normal after percutaneous coronary intervention (PCI). **(A)** Coronary angiogram and **(B)** multiplanar reconstructed image show significant stenosis in the mid left anterior descending artery. Cross-sectional images show **(C)** the proximal reference, **(D)** the culprit lesion, and **(E)** the distal reference. The lesion has no positive remodeling (remodeling index: 0.98), and CT density is 76 HU.

specificity, positive predictive value (PPV) and negative predictive value (NPV), and diagnostic accuracy of the CT characteristics alone or in combination were calculated for prediction of post-PCI cTnT elevation. A p value of <0.05 was considered to indicate statistical significance.

Results

Patient, lesion, and procedural characteristics. Patients were divided into 2 groups according to the presence (group I, n = 36) or absence (group II, n = 71) of post-PCI cTnT elevation 3 times or more (≥3×) the upper limit of normal (0.010 ng/ml) at 24 h after PCI. Baseline patient characteristics are summarized in Table 1. There were no statistically significant differences in the clinical characteristics and medications between the 2 groups. The median post-PCI cTnT levels were significantly higher in patients in group I than in group II (0.088 [0.037 to 0.289] ng/ml vs. 0.010 [0.010 to 0.014] ng/ml, $p < 0.001$). Angiographic findings and procedural results are summarized in Table 2. All lesions were treated with stent implantation. There were no statistically significant differences in target vessels, type B2/C lesions, bifurcation lesions, number of stents implanted, and stent size between the 2 groups. Transient no reflow was observed significantly more frequently in group I than in group II (16% vs. 2%, p = 0.012).

MDCT measurements in the culprit lesion. Representative images from patients in groups I and II are presented in Figures 1 and 2, respectively. Fifty randomly selected lesions were measured by 2 observers. Interobserver and intraobserver measurements of CT attenuation value ($r = 0.92$, $p < 0.0001$, and $r = 0.93$, $p < 0.0001$, respectively) and RI ($r = 0.83$, $p < 0.0001$, and $r = 0.87$, $p < 0.0001$, respectively) were closely correlated, and mean differences in CT attenuation value (0.75 ± 1.58 HU and 2.09 ± 1.31 HU, respectively) and RI (−0.017 ± 0.013 and 0.004 ± 0.011, respectively) were small. Agreement (κ) for the interpretation of spotty calcification was 0.90 and 0.92, respectively. According to the receiver-operating characteristic analysis, the best cutoff points of CT attenuation value was 55 HU, with a sensitivity of 69% and a specificity of 80%, and RI was 1.05, with a sensitivity of 80% and a specificity of 68% for prediction of post-cTnT elevation. Therefore, we defined the CT attenuation value of <55 HU as the lower plaque density and RI of >1.05 as the PR. Lesion characteristics on MDCT are listed in Table 2. The CT attenuation value of the culprit plaque was significantly lower in group I than in group II (43.0 [26.5 to 75.7] HU vs. 94.0 [65.0 to 109.0] HU, $p < 0.001$), and presence of PR (80% vs. 32%, $p < 0.001$) and lesion length (16.7 ± 4.2 mm vs. 14.1 ± 5.1 mm, p = 0.012) were significantly greater in group I than in group II. The prevalence of any coronary plaque subtype and any calcified subtype with the presence or absence of post-PCI cTnT elevation is in Figure 3. Mixed plaques (64% vs. 31%, $p < 0.001$) and spotty calcification (50% vs. 11%, $p < 0.001$) were observed significantly more frequently in group I than in group II, whereas noncalcified plaques (33% vs. 61%, $p < 0.001$) were observed significantly less frequently in group I than in group II. Plaque with ringlike enhancement was observed significantly more frequently in group I than in group II (31% vs. 11%, p = 0.016).

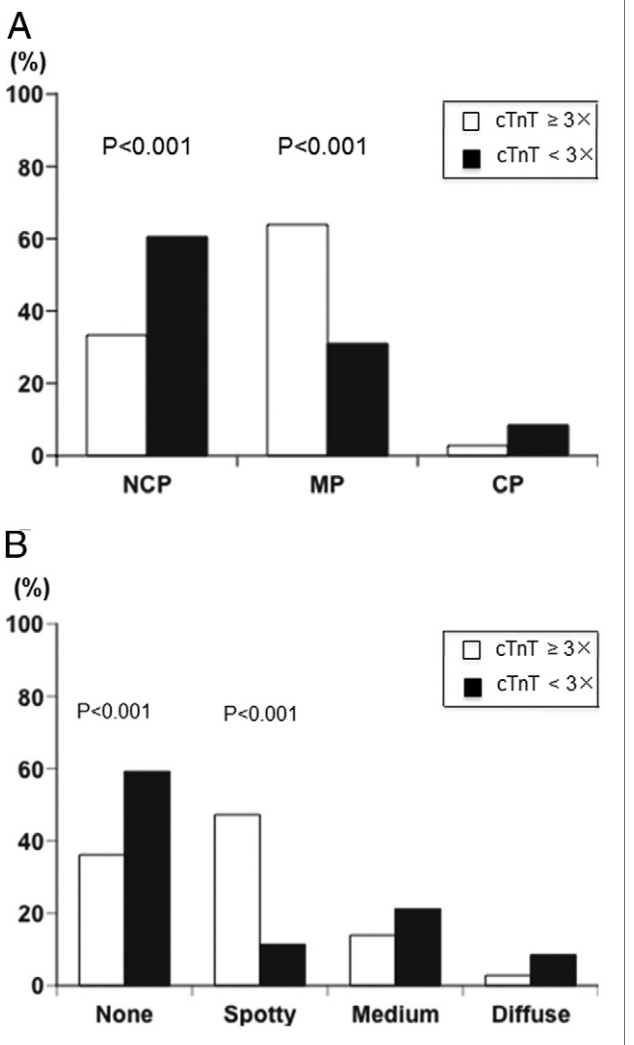

Figure 3 Percentage of Patients With Any Plaque Subtype and Any Calcified Type

Percentage of patients with **(A)** any plaque subtype and **(B)** any calcified type, according to the presence or absence of cardiac troponin T (cTnT) elevation ≥3 times the upper limit of normal (≥3×) after percutaneous coronary intervention (PCI). **Open bars** = cTnT ≥3×; **solid bars** = cTnT <3×. CP = calcified plaque; MP = mixed plaque; NCP = noncalcified plaque.

Univariate and multivariate analysis for predicting post-PCI cTnT elevation. Post-PCI cTnT elevation was observed in 36 of the 107 patients (33.6%) in this study. According to the univariate analysis (Table 3), lesion length, PR, CT attenuation, and spotty calcification were significantly associated with post-PCI cTnT elevation. Multiple logistic regression analysis showed that the

Table 3 Univariate and Multivariate Logistic Regression Analyses for Prediction of Cardiac Troponin Elevation

	Univariate Analysis			Multivariate Analysis		
Factor	OR	95% CI	p Value	OR	95% CI	p Value
Age	1.03	0.98–1.08	0.161	1.02	0.96–1.07	0.522
EEM CSA	1.04	0.96–1.12	0.272			
Lesion length	1.12	1.02–1.23	0.012	1.07	0.97–1.20	0.187
RI >1.05	8.65	3.45–24.2	<0.001	4.54	1.36–15.9	0.014
CT value <55 HU	9.25	3.79–24.1	<0.001	2.03	0.57–7.05	0.265
Spotty calcification	7.04	2.71–19.7	<0.001	4.27	1.30–14.8	0.016
Log CRP	1.29	0.48–1.22	0.271			
Statin use	0.57	0.22–1.44	0.237			

CI = confidence interval; CRP = C-reactive protein; OR = odds ratio; RI = remodeling index; other abbreviations as in Table 2.

presence of PR (odds ratio: 4.54; 95% confidence interval: 1.36 to 15.9; p = 0.014) and spotty calcification (odds ratio: 4.27; 95% confidence interval: 1.30 to 14.8; p = 0.016) were statistically significant independent predictors for post-PCI cTnT elevation after adjustment for multiple confounders.

Diagnostic accuracy of CT characteristics for predicting post-PCI cTnT elevation. The sensitivity, specificity, PPV and NPV, and diagnostic accuracy of each CT characteristic alone or in combination are shown in Table 4. Presence of all 3 characteristics (CT <55 HU, RI >1.05, and spotty calcification) showed a high PPV of 94%, and absence of all 3 showed a high NPV of 90% for predictive value of cTnT elevation.

Discussion

Importantly, our study demonstrated a direct relation between pre-PCI plaque composition by MDCT and post-procedural myocardial injury/infarction in SAP patients. Post-PCI cTnT elevation ≥3× the upper limit of normal was observed in 33.6% of patients with SAP. The presence of positive remodeling and spotty calcification were the significant predictors of post-PCI cTnT elevation. For prediction of post-PCI cTnT elevation, the presence of all 3 CT characteristics showed a high PPV of 94%, and their absence offered a high NPV of 90% for the exclusion of culprit lesions by noninvasive MDCT imaging.

Relation between plaque characteristics on MDCT and cTnT elevation. Cardiac biomarker cTnT is sensitive and specific for detection of myocardial damage. Porto et al. (22) found that the cause of periprocedural myocardial necrosis after PCI was the impairment of flow in coronary side branches and distal embolization of atheromatous or thrombotic materials. Therefore, pre-PCI plaque composition may have an impact on myocardial injury/infarction during PCI. However, there are few published data regarding the relation between pre-PCI plaque composition by MDCT and post-PCI cardiac biomarker levels. Nakazawa et al. (23) reported that patients who experienced transient no reflow during PCI had lower plaque CT density values in culprit lesions. Uetani et al. (12) demonstrated that post-procedural myocardial injury was associated with the volume and fraction of low-attenuation plaques by MDCT. In the present study, CT attenuation value of <55 HU was associated with post-PCI cTnT elevation. While in earlier studies, a mean CT density of 14 to 47 HU was found in lipid-rich plaque (8,24,25), Pohle et al. (26) showed a mean density of 58 HU (median 53), and Leber et al. (16) reported that a low CT density value (49 ± 22 HU) is considered to correspond to soft plaque identified on IVUS. This difference most likely results from the natural course of atherosclerotic plaque or slice thickness and contrast medium concentration that affect plaque density measurements (27,28). It will be possible to use our cutoff point of CT attenuation value <55 HU for prediction of post-PCI cTnT elevation clinically.

Table 4 Diagnostic Accuracy of CT Characteristics for Prediction of Cardiac Troponin Elevation

Factor	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
CT value <55 HU	69	80	64	84	77
RI >1.05	80	68	56	87	72
Spotty calcification	50	88	69	78	76
CT value <55 HU + RI >1.05	67	85	69	83	79
CT value <55 HU + RI >1.05 + spotty calcification	47	98	94	79	81
CT value <55 HU or RI >1.05	83	63	54	88	70
CT value <55 HU or RI >1.05 or spotty calcification	86	58	52	90	68

NPV = negative predictive value; PPV = positive predictive value; other abbreviations as in Tables 2 and 3.

Predictive value for detection of cTnT elevation. In the present study, PR and spotty calcification were significant predictors of post-PCI cTnT elevation. Furthermore, presence of all 3 CT characteristics (CT attenuation value <55 HU, RI >1.05, and spotty calcification) showed a high PPV of 94%, and their absence showed a high NPV of 90%. The PR has been associated with plaque vulnerability in clinical studies. A previous report of an IVUS study confirmed that PR (RI >1.05) was an important factor in the culprit lesion (29). Histopathological characteristics of ruptured plaques showed the same characteristics of the vulnerable plaques. Plaque rupture occurred not only in patients with unstable angina pectoris or myocardial infarction but also in those with SAP. The rupture site has larger arterial wall and lumen areas and more PR (30). Another histopathological report showed that coronary plaques with PR had a higher lipid content and macrophage count (31), which indicated plaque vulnerability. Ehara et al. (32) found by IVUS study that small calcium deposits were present with significant frequency in the culprit lesion segments in patients with ACS. Burke et al. (33) reported that autopsy data showed the degree of calcification was greatest for acute and healed plaque ruptures and the least for plaque erosion. Calcification in coronary atherosclerosis is associated with 1 type of plaque instability, namely, plaque rupture (33).

The data in the present study showed that some SAP patients have spotty calcification and that plaque with spotty calcification was 1 of the predictors of post-PCI cTnT elevation. A previous MDCT study showed that mixed lesions were associated with adverse events on follow-up (13). Thin-cap fibroatheromas on virtual histology IVUS were most prevalent in mixed plaques, suggesting a higher degree of vulnerability of these mixed plaques on MDCT (14). In the present study, we found that mixed plaques with low-attenuation plaques, PR, and spotty calcification have the potential to predict an elevation of cTnT after PCI. We also demonstrated that none of the conventional risk factors allowed differentiation between the post-PCI cTnT elevation and the elevation-free group. These findings were in agreement with a previous MDCT study by Motoyama et al. (11), and were also supported by recent near-infrared spectroscopy data (34).

Clinical implications. These data suggest that it would be worthwhile to consider the identification of these vulnerable plaques by MDCT before PCI. If the plaque has the characteristics of low plaque density, PR, and spotty calcification, a plan for prevention of post-PCI cTnT elevation can be made before the PCI procedure. Further efforts to minimize procedural events may require aggressive attempts to prevent distal embolization (35). As for medications, Kawai et al. (36) reported that intravenous administration of nicorandil before PCI can prevent slow coronary flow phenomenon. A single high loading dose (80 mg) of atorvastatin administered within 24 h of PCI reduces the incidence of periprocedural MI in elective PCI (37). Therefore, the use of these medicines during or before the procedure might the possibility of preventing post-procedural myocardial injury/infarction in the perioperative period.

Study limitations. First, we acknowledge that because of the relatively small sample size in the present study, our findings should be confirmed in a larger number of patients. Second, we excluded patients with severely calcified lesions in this study. In general, SAP patients frequently have more severe coronary artery calcification in their coronary arteries than do ACS patients. If we included these patients in this study, it is probable that the occurrence of post-PCI cTnT elevation would be lower than that of our presented data. Third, CT plaque density can be altered by the concentration of the contrast agent administered or by contrast type, regardless of the presence or absence of calcification near the plaque and of the model of CT used. Finally, we consider it necessary to investigate the relation between the MDCT image and histopathology and to confirm the correspondence between these imaging characteristics and histopathologic findings.

Conclusions

The present MDCT study showed that post-PCI cTnT elevation occurred in lesions with low-attenuation plaques, PR, and spotty calcification in patients with SAP. MDCT may play an important role in detecting which lesions are at high risk for myocardial necrosis after PCI.

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